Orofacial Pain

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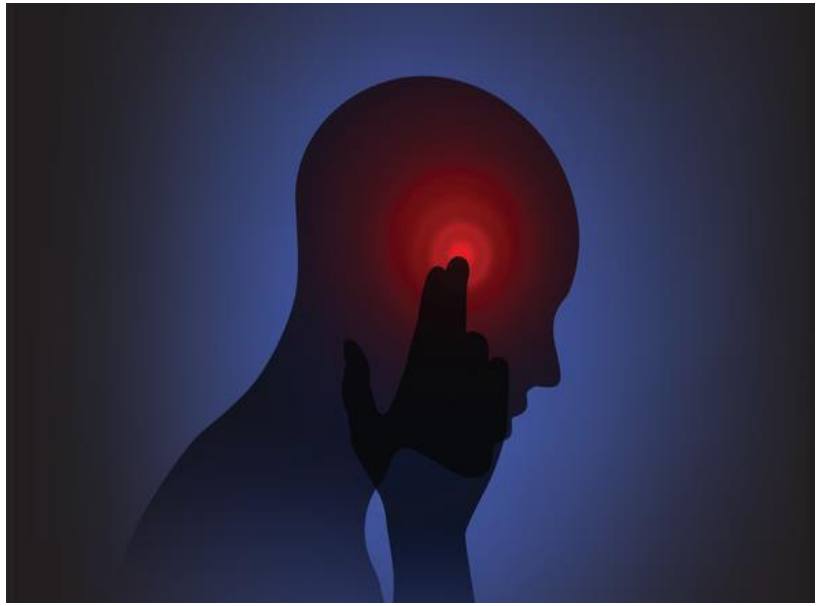
Second Course Lec-1



Pain has been defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage".

Orofacial pain (OFP) prevalent in the general population; around 23%,

of which 7%–11% is chronic.

Acute OFP is primarily associated with the teeth and their supporting structures.

Most frequently, dental pain is due to dental caries, although a broken filling or tooth-abrasion may also cause dental sensitivity.

Other oral pains are usually periodontal or gingival in origin.

Chronic orofacial pain (COFP)

is a term used to describe painful regional syndromes with a chronic, unremitting pattern.

Anatomic consideration

. Cranial nerve V (CN V), the trigeminal nerve, is the dominant nerve that relays sensory impulses from the orofacial area to the central nervous system

The facial (CN VII), glossopharyngeal (CN IX), and vagus (CN X) nerves and the upper cervical nerves (C2 and C3) also relay sensory information from the face and surrounding area.

Clinically COFP may be subdivided into three main symptomatic classes

1- Musculoskeletal, entities are dealt with "Temporomandibular Disorders."

2-Neurovascular.

3- Neuropathic

Diagnostic Tests:

Any test to select is guided by history & physical examination:

1. CT and/or MRI (to rule out intracranial pathology)
2. TMJ radiography
3. Diagnostic occlusal appliance
4. Cervical spine films
5. Lab. (ESR ,c- reactive protein)
6. Biopsy
7. VAS (visual analog scale)

CHRONIC OROFACIAL PAIN

A-Neurovascular Pain

Neurovascular pains include migraines and the trigeminal autonomic cephalgias (TACs) include cluster headache

1-MIGRAINE Headache

Migraine are moderate to severe pain, which is usually throbbing and often unilateral >bilateral (frontal, temporal, occipital) and intraorally referral to maxillary sinus associated features of nausea, sensitivity to light, sound, and exacerbation with head movement. Last 4–72 h. May wake from sleep—usually in early morning.

Migraine is more common in women.

It may bepreceded by an aura of neurological dysfunction, such as visual disturbances, vertigo,numbness, or weakness. The pain may be moderate or incapacitating.

In many patients, migraine is triggered by specific

factors, such as menses, weather changes, irregular sleep, alcohol, or certain foods.

The clinical features of migraine are separated into two types of headache:

migraine without aura (common migraine) and

migraine with aura (classic migraine).

migraine aura without headache, also known as silent migraine – where an aura or other migraine symptoms are experienced, but a headache does not develop

Causes of migraines:

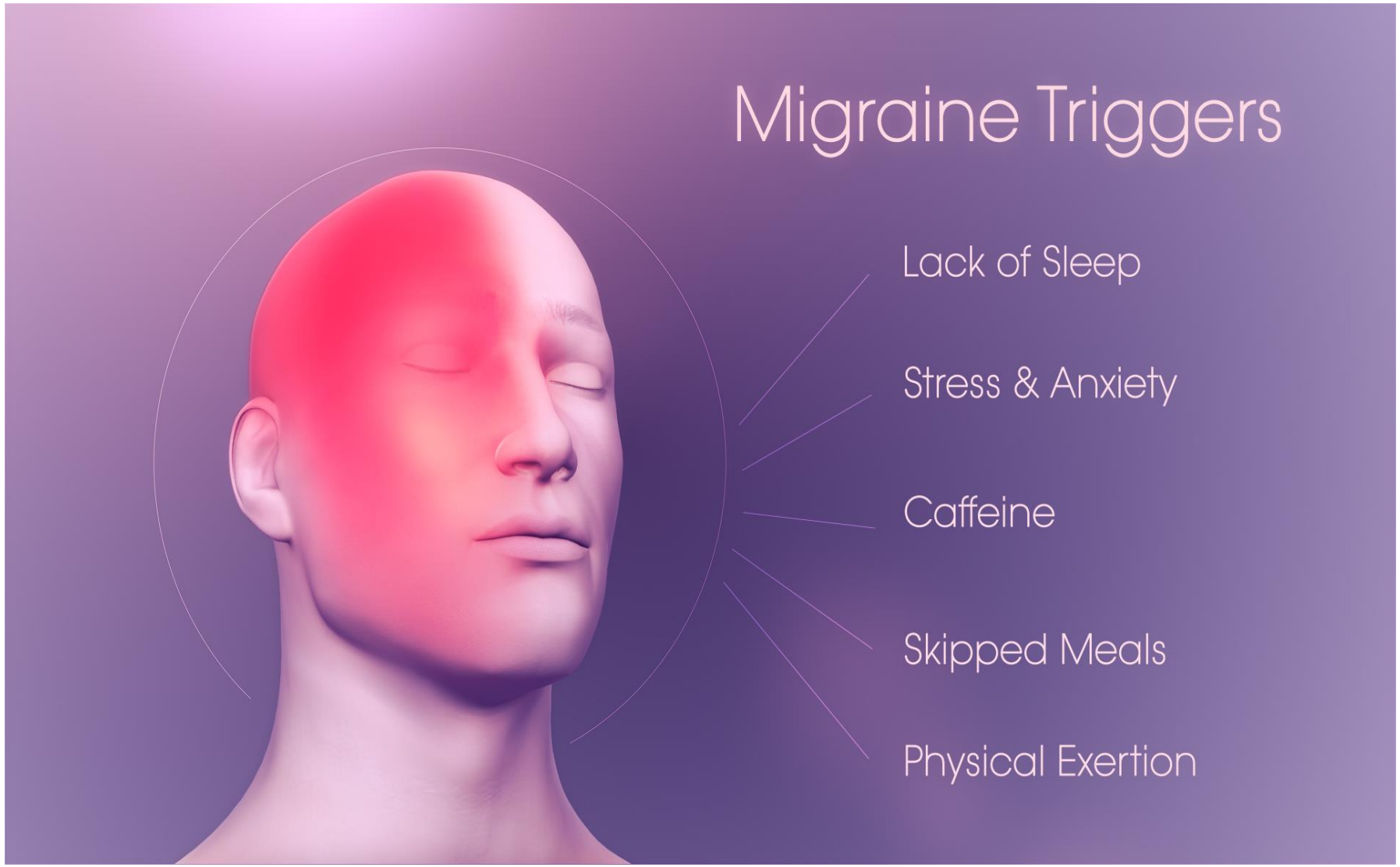
The exact cause of migraines is unknown, although they're thought to be the result of temporary changes in

the chemicals, nerves and blood vessels in the brain.

Around half of all people who experience migraines also have a close relative with the condition, suggesting that genes may play a role.

Some people find migraine attacks are associated with certain triggers, which can include:

- Starting their period
- Stress
- Tiredness
- Certain foods or drinks (e.g. Caffeine
- Skipped of meals
- Physical Exertion


Migraine Triggers
Lack of Sleep
Stress & Anxiety
Caffeine
Skipped Meals
Physical Exertion

Etiology and Pathogenesis

Many theories that explain that and the most common is

Vascular Theory

The aura of migraine was once thought to be caused by cerebral vasoconstriction and the headache by reactive vasodilation, which explained the throbbing quality of migraine and the relief of pain by ergots. It is now believed that the aura is caused by

neuronal dysfunction rather than ischemia.

Treating migraines:

There's no cure for migraines, but a number of treatments are available to help reduce the symptoms,these include:

- painkillers – including over-the-counter medicines like paracetamol and ibuprofen
- triptans – medicines that can help reverse the changes in the brain that may cause migraines
- anti-emetics – medicines often used to help relieve people's feeling of sickness (nausea) or being sick During an attack, many people find that sleeping or lying in a darkened room can help.

Drugs that are useful in aborting migraine include ergotamine and sumatriptan, which can be given orally, nasally, rectally or parenterally.

Ergotamine Initial dose: Oral, Sublingual: 2 mg ergotamine in fixed combination with caffeine given as quickly as possible after the first symptom of headache.
Additional 1 mg doses can be given every 30 minutes until the headache has been aborted or until a total dose of 6 mg has been reached or 10 mg/week.

These drugs must be used cautiously since they may cause hypertension and other cardiovascular complications.

Preventing migraines

If you suspect a specific trigger is causing your migraines, such as stress or a certain type of food,

avoiding this trigger may help reduce your risk of experiencing migraines.

It may also help to maintain a generally healthy lifestyle, including regular exercise, sleep and meals, as well as ensuring you stay well hydrated and limiting your intake of caffeine and alcohol. If your migraines are severe or you have tried avoiding possible triggers and are still experiencing symptoms, a GP may prescribe medicines to help prevent further attacks.

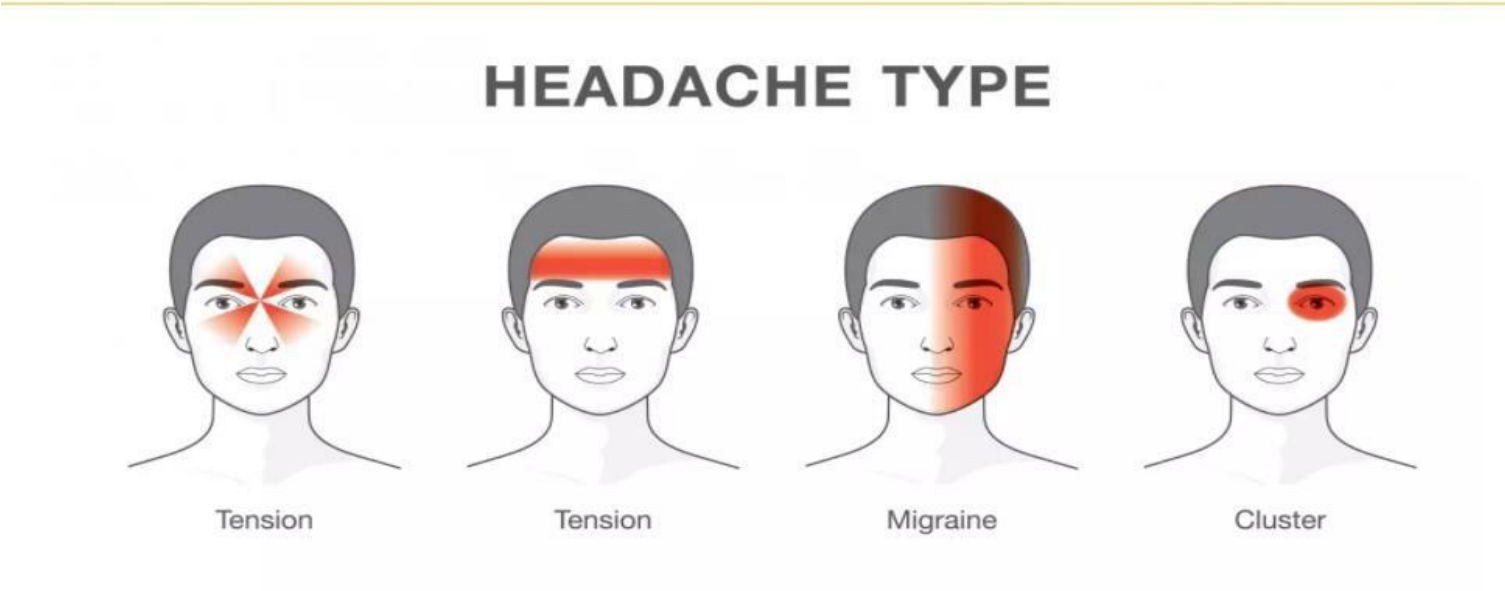
Medicines used to prevent migraines include the anti-seizure medicine topiramate and a medicine called propranolol that's usually used to treat high blood pressure. It may take several weeks before your migraine symptoms begin to improve.

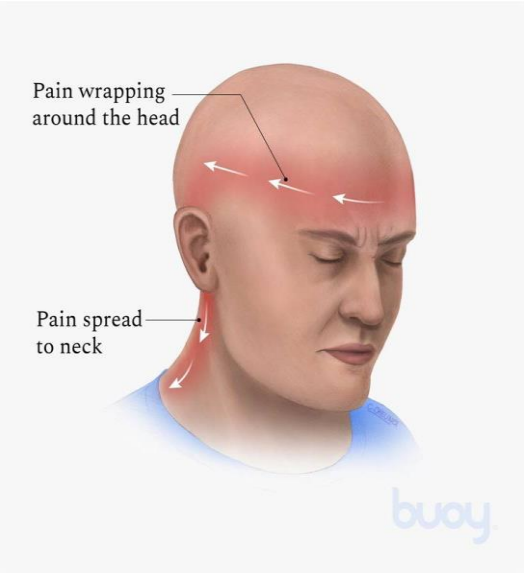
2- TENSION-TYPE HEADACHES (TTH)

Over 80% of adults experience TTH, characterized by bilateral, tight, band-like discomfort periodically, and it is common in both adolescents and children. Women tend to be affected more than men.

The clinical presenting symptoms and signs of TTH is chronic daily headache include bilateral, band-like tightness and pressure with beginning usually in late morning , poor appetite, restlessness. Unlike migraine, nausea and vomiting are rare occurring only in cases of severe pain in a minority of patients.

Tension-type headache is most commonly self-treated with NSAIDs and acetaminophen. Psychological management includes simple counseling and hypnosis, whereas relaxation and biofeedback measures, including acupuncture, are helpful physiologic treatments





3- Cluster Headache (CH)

Pain in CH is usually severe, periorbital or ocular, but varies. About 80% of CH patients are heavy smokers, and 50% have a history of heavy ethanol use. Typically appears between the ages of 20–29 years, is more common than previously thought, and seems to affect men more than women.

Attacks last from 30 minutes to 2 hours and have a nocturnal onset in half of cases, waking patients several hours after falling asleep. Pain is commonly associated with autonomic symptoms, including ipsilateral lacrimation, reddening of the eye, nasal stuffiness, and nausea.

In "upper CH" the forehead, temporal, and parietal regions are involved, whereas in

"lower CH" the temporal and sub occipital regions are affected with radiation to the teeth, jaws, neck, and cheeks.

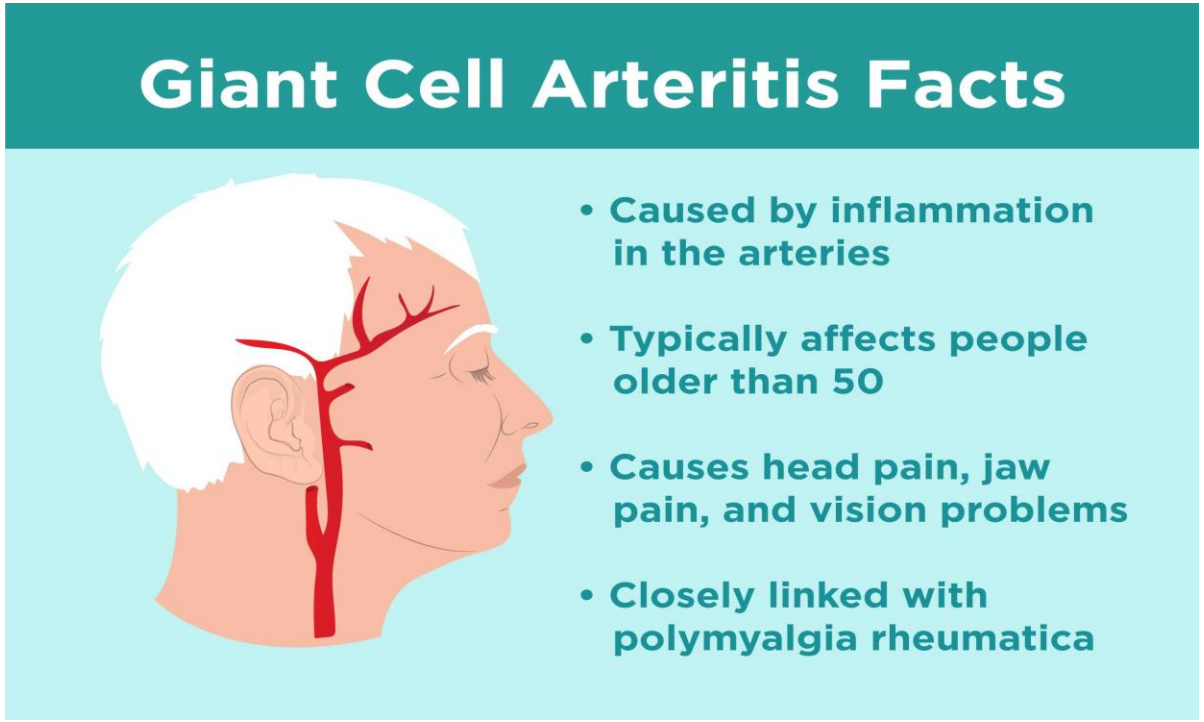
Pain is unilateral and in 20% of cases may change sides.

Oxygen inhalation (9 L/min) over a period of 20 minutes results in rapid resolution of symptoms in over 70% of cases. Intranasal sprayed lidocaine 4% is also effective in nearly half of cases.

4- TEMPORALARTERITIS

Temporal (or giant cell) arteritis is a systemic inflammatory disorder that often involves the **extracranial carotid circulation**. Symptoms of diffuse unilateral headache (along with chest pain, jaw pain , claudication, fever, and weight loss and elevated erythrocyte sedimentation (ESR).

Medical care for temporal arteritis (TA) is supportive and symptom specific, Glucocorticoid therapy.



B--Neuropathic Orofacial Pain

Neuropathic OFP includes a number of clinical entities; are Trigeminal Neuralgia (TN), glossopharyngeal neuralgia (GN) , burning mouth syndrome (BMS), facial post herpetic neuropathy and painful posttraumatic trigeminal neuralgia.

1- Trigeminal Neuralgia (TN)

TN is an excruciating, short-lasting (from a fraction of a second up to 2 minutes), or electrical most commonly unilateral facial pain in both the maxillary and mandibular branches of the trigeminal nerve, unilateral and pain radiation is generally within the dermatome of the origin. The most common is the classical unrelated to pathology and most probably caused by neurovascular compression of the trigeminal nerve root.

Most paroxysms occur during waking hours, but may awaken the patient. Typically pain is precipitated by light, innocuous touch at sites called “trigger areas". Trigger factors" such as noise, lights, and stress may also induce pain.

Diagnosis may done by MRI may show neurovascular contact at the CN5 nerve root. TN may also occur secondary to tumors and multiple sclerosis.
Carbamazepine remains the drug of choice for TN treatment (100–200 mg twice daily of the slow release formulation increase as needed), or Oxcarbazepine (300 mg × 3/d, titrate as needed). Gabapentin may be useful in selected TN cases (200–300 mg × 2/d).

Surgical (best prognosis in typical TN early after onset):

•• Peripheral level

•• Ganglion level

•• Trigeminal root level

2- Glossopharyngeal Neuralgia

The glossopharyngeal (IX) nerve has two main sensory branches:

1- The auricular (tympanic)

2- The pharyngeal.

In pharyngeal-GN, the pharynx or posterior tongue-base are involved. Pain radiates to the inner ear or the angle of the mandible, and may include the eye, nose, maxilla, or shoulder and even the tip of the tongue. In tympanic-GN, pain predominates in the ear but may radiate to the pharynx.

GN is a paroxysmal, unilateral, severe pain that is sharp, stabbing, shooting, or lancinating. Pain intensity is usually milder than TN but may vary and attacks last from a fraction of a second up to 2 minutes.

Trigger areas are located in the tonsillar region and posterior pharynx, and these display a refractory period. Swallowing, chewing, talking, coughing and/or yawning, sneezing, clearing the throat, and rubbing the ear activate these areas.

Carbamazepine is usually successful and is the favored medication. Alternatives include Baclofen, oxcarbazepine, gabapentin, and phenytoin.

3- Burning Mouth Syndrome (Glossodynia)

BMS is a poorly understood pain condition that is most probably neuropathic. The condition is also known as stomatodynia and is characterized by a burning mucosal pain with no significant physical signs and is common in postmenopausal women.

BMS may be sub classified into "primary" or idiopathic BMS for which a neuropathological cause is likely and cannot be attributed to any systemic or local cause and

"secondary BMS" (SBMS) resulting from local or systemic pathological conditions.

The "primary" or idiopathic BMS symptoms do not follow anatomic pathways, there are no mucosal lesions or known neurologic disorders to explain the symptoms, and there are no characteristic laboratory abnormalities.

ETIOLOGY AND PATHOGENESIS

The cause of primary BMS remains unknown, but a number of factors have been suspected

1. including hormonal changes.
2. salivary gland hypofunction.
3. chronic low-grade trauma.
4. Psychiatric abnormalities.

The increased incidence of BMS in women after menopause has led investigators to suspect an **association with hormonal changes**

Local factors and diseases known to induce SBMS include :

oral candidiasis, lichen planus, and allergies.

While , Systemic disorders that induce SBMS include

hormonal changes, deficiencies of vitamin B12, folic acid or iron, diabetes mellitus, side

effects of medications, and autoimmune diseases.

CLINICAL MANIFESTATIONS

The primary location of the burning complaint is the tongue, usually the anterior 2/3. However, usually more than one site is involved and in addition to the tongue, hard palate, lips, and gingivae are frequently involved. Pain is most commonly described as burning or hot and intensity varies from mild to severe. BMS is typically of spontaneous onset and lasts from months to several years.

The pain pattern may be irregular, but some patients may complain that pain increases toward the end of the day, partial remission has been reported in about one half to two-thirds of patients, six to seven years after onset. Spontaneous remission is very rare.

Women experience symptoms of BMS seven times more frequently than men. When questioned, 10 to 15%

of postmenopausal women are found

TREATMENT

Once the diagnosis of BMS has been made by eliminating the possibility of detectable lesions or underlying medical disorders, the patient should be reassured of the benign nature of the symptoms. Counseling (advising) the patient in regard to the nature of BMS is helpful in management, particularly because many patients will have had multiple clinical evaluations without an explanation for the symptoms. Topical therapies may be effective and are useful in elderly, medically compromised patients. The most established is clonazepam (1 mg) which

should be sucked and subsequently spat out three times daily. Topical anesthetics may decrease or increase

pain and are therefore unpredictable. The drug therapies that have been found to be the most helpful are low doses of TCAs, such as amitriptyline and doxepin, or clonazepam (a benzodiazepine derivative). It should be stressed to the patient that these drugs are being used not to manage psychiatric illness, but for their well- documented analgesic effect.

4- POSTHERPETIC NEURALGIA (PHN)

PHN is a dermatomal disease persisting or recurring ≥3 months after the acute HZ stage. PHN rarely affects the trigeminal nerve and when it does it is mostly in the ophthalmic branch which accounts for 22% of PHN patients.

Pain quality is burning, throbbing, stabbing, shooting, or sharp. Itching is very common and prominent in trigeminal dermatomes and may be subjectively graded as worse than pain. Patients with PHN experience persistent pain, paresthesia, hyperesthesia, and allodynia months to years after the zoster lesions have healed.

***Etiology and Pathogenesis*.** Herpes zoster (shingles), is caused by the reactivation of latent varicella-zoster virus infection that results in both pain and vesicular lesions along the course of the affected nerve. Approximately 15 to 20% of cases of herpes zoster involve the trigeminal nerve although the majority of these cases affect the ophthalmic division of the fifth nerve, resulting in pain and lesions in the region of the eyes and forehead. Herpes zoster of the maxillary and mandibular divisions is a cause of facial and oral pain as well as of lesions. In a majority of cases, the pain of herpes zoster resolves within a month after the lesions heal.

The pain and numbness of PHN results from a combination of both central and peripheral

mechanisms The varicella-zoster virus injures the peripheral nerve by demyelination , wallerian degeneration, and sclerosis, but changes in the CNS, including atrophy of dorsal horn cells in the spinal cord, have also been associated with PHN.

Management.

Many treatment options are available for the management of PHN, and the method chosen should depend on the severity of the symptoms as well as the general medical status of the patient.

Treatment includes topical and systemic, drug therapy and surgery.

Topical therapy includes the use of topical anesthetic agents, such as lidocaine, or analgesics, particularly capsaicin. Lidocaine used either topically or injected gives short-term relief from severe pain. Capsaicin,

5- Painful Posttraumatic Trigeminal Neuropathy

The term painful posttraumatic trigeminal neuropathy (PTTN) is novel and has recently been adopted by the International Headache Society (IHS). Some patients develop chronic pain following negligible nerve trauma such as root canal therapy or following considerable injury to nerve bundles, such as in fractures of the facial skeleton.

Following dental implant surgery 1%–8% and following orthognathic jaw surgery 5%–30% of patients may remain with permanent sensory dysfunction but the incidence of chronic pain is unclear.